



Biotechnological Mitigation of Phthalates in the Food Chain: Packaging Migration, Dietary Exposure, and Probiotic Detoxification

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Abstract

Background and aims: Phthalates are plasticizers produced in volumes so large that dietary exposure has become universal. Because they remain unbound within polymer networks, these compounds migrate into food during processing, storage, and distribution, with additional inputs entering crops and livestock through environmental contamination.

Materials and Methods: Following a structured search across four databases, this review synthesizes evidence on the routes of food contamination, analytical challenges, dietary intakes, toxicological endpoints, and emerging mitigation strategies.

Results and Conclusion: Occurrence data consistently show that lipid-rich items such as edible oils and dairy products accumulate the heaviest loads, though storage temperature and acidity can also drive measurable leaching into bottled water and soft drinks. Estimated intakes indicate that young children and high consumers of packaged foods absorb a noticeable share of established tolerable daily intakes, particularly when compounds are assessed as cumulative mixtures under European Food Safety Authority guidance. Given that current regulations and industrial substitutes like DINCH and DEHT leave historical residues in circulation, biological mitigation has emerged as a complementary safeguard. Food-grade lactic acid bacteria, notably strains of *Lactobacillus* and *Lactiplantibacillus*, can physically sequester phthalate diesters and reactive monoesters such as MEHP through peptidoglycan and exopolysaccharide surface binding, reducing free toxicant fractions in fermented matrices and accelerating fecal clearance in vivo. We conclude by outlining critical research priorities, including outdated occurrence surveys, single-compound regulatory baselines, and the need to validate microbial biosorption under commercial food-processing conditions.

What is “already known”:

- Phthalates are not covalently bound to polymer matrices, so they migrate from packaging into food, particularly lipid-rich products.
- Dietary exposure to phthalates is nearly universal, and phthalates act as endocrine-disrupting chemicals.
- Regulations restrict only some phthalates, and substitute plasticizers do not remove historical residues from circulation.

What this article adds:

- An integrated account connecting sources of food contamination (packaging, processing equipment, and the environment), migration mechanisms, and measured occurrence in mineral water, soft drinks, dairy products, and edible oils.
- Estimated dietary intakes compared with toxicological reference values, including the EFSA group-TDI, showing that safety margins are smaller for children and high consumers.
- LAB can sequester phthalates through surface binding and accelerate their fecal clearance, as a complementary mitigation strategy.

1. INTRODUCTION

Phthalates, the diesters of phthalic acid, belong to the synthetic chemicals produced in the largest volumes worldwide. Since the 1920s they have served above all as plasticizers for polyvinyl chloride (PVC), and consumption in the United States alone has been put at more than 340 million pounds per year [1]. Their uses extend well beyond plastics. Perfumes, lotions, pharmaceuticals, adhesives, and printing inks all rely on them as solvents, fixatives, or coating agents [2].

This widespread use has a downside. Phthalates are not covalently bound to the polymer matrices in which they are embedded, so they migrate readily out of products into the surrounding environment, including air, dust, water, and, most importantly for public health, food [3]. Once absorbed, they and their metabolites act as endocrine-disrupting chemicals, interfering with hormone synthesis, receptor binding, and signaling pathways in ways that affect reproductive development, metabolic regulation, and, in some cases, red blood cell integrity [1, 4]. Concerns of this kind are not purely theoretical. Recent reviews have linked dietary phthalate and bisphenol exposure to early-onset obesity and precocious puberty in children, two conditions whose prevalence keeps climbing [5]. Findings like these reinforce the view that diet, rather than occupational or indoor-air contact, is the route through which most people encounter these compounds on a daily basis.

Regulators have responded to the evidence, though unevenly. Several high-production phthalates have been banned or restricted in toys and childcare articles, and the European Food Safety Authority re-examined the phthalates used in food-contact materials in 2019, confirming the earlier tolerable

daily intakes and introducing a group-TDI to account for their combined action [49]. Restrictions, however, cover only part of the compounds on the market, and packaging formulations change faster than the rules that govern them [6]. Phthalate metabolites nevertheless continue to appear in the urine of the vast majority of biomonitored populations [7], which suggests that current controls have reduced dietary exposure without eliminating it.

Earlier reviews of phthalates in food have either concentrated on a single food category or predate the regulatory and analytical developments of the last five years [7]. What is missing is an integrated account that connects the sources of contamination, the mechanisms of migration, and the measured occurrence in foods people actually eat. The present review was written to fill that gap. It summarizes the classification and industrial applications of the phthalates most relevant to food safety, describes how packaging materials, processing equipment, and environmental contamination introduce these compounds into food, and then assembles recent occurrence and exposure data for four major categories, namely mineral water, soft drinks, dairy products, and edible oils and fats. Where data permit, estimated intakes are set against toxicological reference values. We also examine biological mitigation using lactic acid bacteria, and identify the research gaps that still limit our understanding.

2. MATERIALS AND METHODS

2.1. Literature Search Strategy

A comprehensive literature search was conducted across four electronic databases: PubMed, Scopus, Web of Science (Core Collection), and Google Scholar. The search covered publications from January 1990 through May 2026, with the final update executed on



May 20, 2026. Search algorithms combined controlled vocabulary and free-text terms using Boolean logic: ("phthalate" OR "phthalate ester" OR "phthalic acid ester" OR "PAE" OR "DEHP" OR "DBP") AND ("food packaging" OR "food contact materials" OR "migration" OR "dietary exposure" OR "dairy" OR "edible oil" OR "mineral water" OR "soft drinks") AND ("lactic acid bacteria" OR "probiotics" OR "Lactobacillus" OR "Lactiplantibacillus" OR "biosorption" OR "biodegradation"). In addition, backward and forward citation tracking (snowballing) was performed on retrieved review articles and authoritative regulatory assessments (e.g., EFSA and US FDA opinions) to capture historically important datasets that might have been missed by database indexing.

2.2. Eligibility Criteria

Studies were evaluated against predetermined eligibility criteria. Inclusion criteria required that publications: (i) report original quantitative data on phthalate occurrence, migration kinetics, or dietary intake within the targeted food matrices (mineral water, soft drinks, dairy products, and edible oils); (ii) investigate biological binding, biosorption, or mitigation mechanisms by food-grade microorganisms or characterized probiotic strains; (iii) be published in peer-reviewed English-language journals or represent official reports from recognized food safety agencies; and (iv) provide documented analytical methodologies (e.g., GC-MS, LC-MS/MS, or SPME) with acceptable quality-control parameters. Exclusion criteria eliminated: (i) investigations confined strictly to non-dietary matrices (e.g., indoor air, house dust, cosmetics, toys, medical tubing, or occupational factory settings) without a direct dietary link; (ii) conference abstracts, non-peer-reviewed preprints, patents, and commercial trade reports; (iii) environmental remediation studies focusing on soil sludge or industrial wastewater treatment that lacked relevance to food systems; and (iv) studies lacking procedural blank controls or baseline analytical validation.

2.3. Data Extraction and Organization

The study selection process followed a structured screening sequence. Initial database searches identified 642 records. Following automated and manual deduplication, 418 unique titles and abstracts were screened. Of these, 287 records were excluded as irrelevant to the core matrix or mechanistic scope. The remaining 131 full-text publications and regulatory documents were evaluated against the eligibility criteria, leading to the selection of 62 peer-reviewed articles and official agency reports for detailed thematic synthesis. For each selected study, standardized data were extracted: target phthalate congeners, food matrix or food simulant, contact duration and temperature, sample origin, analytical instrumentation, quality assurance protocols (procedural blank controls and recoveries), and measured concentration ranges. For biological mitigation studies, specific microbial strains, cell-wall fractions, binding efficiencies, and operational conditions (pH, contact time, cell viability) were recorded. Extracted findings were synthesized into five thematic sections: classification and properties, contamination routes, occurrence across food groups, probiotic-mediated biosorption, and current regulatory and research gaps.

3. CHEMICAL STRUCTURE, CLASSIFICATION, AND PHYSICO-CHEMICAL PROPERTIES OF PHTHALATES

Chemically, phthalates are diesters of phthalic acid, 1,2-benzenedicarboxylic acid, to use its formal name. Making them is not complicated: phthalic anhydride reacts with an aliphatic alcohol, and that choice of alcohol ends up controlling most of what matters about the final molecule, how well it dissolves in water, how volatile it is, how much it prefers fat over water. A lot rides on one variable. That is part of why the phthalate family has grown so large, and why two compounds from the same family can behave in completely different ways once they end up near food [8, 9].

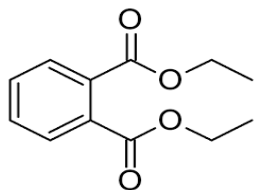
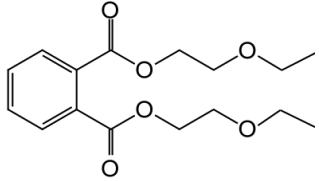
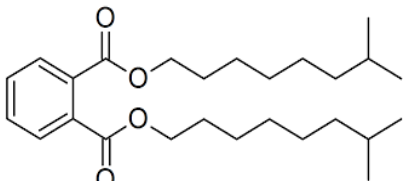
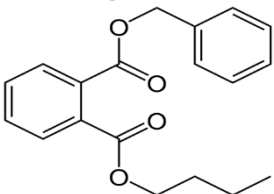
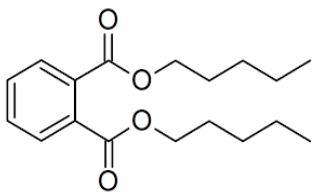
3.1. Classification and Main Applications

The usual way to sort phthalates is by the length of their ester side chains. High-molecular-weight phthalates, or HMW phthalates, have alkyl chains

running seven to thirteen carbons long. Di(2-ethylhexyl) phthalate (DEHP), diisononyl phthalate (DINP), and di-n-octyl phthalate (DnOP) fall into this group and are, by some distance, the most commonly encountered. Their main use is flexible vinyl; polyvinyl chloride (PVC) plasticized with these compounds shows up in food packaging, in flooring and wall coverings, in medical devices [8, 9]. Low-molecular-weight phthalates are a different story. With chains of just three to six carbons, diethyl

phthalate (DEP) and di-n-butyl phthalate (DBP) rarely make it into plastic food packaging at all; instead they turn up as solvents in perfumes and lotions, as plasticizers for cellulose acetate, and in lacquers and coatings, including some designed to control drug release in pharmaceuticals [2]. The structures, applications, and reported toxic effects of the phthalates covered here are summarized in Table 1.

Table 1. Chemical structures, principal applications, and reported toxic effects of the phthalates most relevant to food safety

Phthalate Name & Identifiers	Chemical Structure	Application	Toxic Effect
Diethyl Phthalate (DEP) CAS: 84-66-2 MW: 222.24 g/mol Log Kow: 2.47		• Plasticizers • Cosmetic • Fragrances • Coating • Pesticides	• Hepatotoxicity • Male reproductive toxicity
Di(2-ethylhexyl) phthalate (DEHP) CAS: 117-81-7 MW: 390.56 g/mol Log Kow: 7.50		• Food Packaging • Medical Tubes • Electrical products • Paint and Coatings • Children's Dolls • Plasticizers • Drinking Straws • Adhesive and Sealants	• Hepatotoxicity • Cytotoxicity • Reproductive toxicity
Diisononyl phthalate (DINP) CAS: 28553-12-0 MW: 418.61 g/mol Log Kow: 8.80		• Plasticizers • Drinking Straws • Adhesive and Sealants	• Hepatotoxicity • Nephrotoxicity
Benzyl butyl phthalate (BBP) CAS: 85-68-7 MW: 312.36 g/mol Log Kow: 4.59		• Food packaging • Food Conveyor Belts • Adhesive and Sealants • Industrial Solvent	• Cytotoxicity • Developmental toxicity
Di-n-butyl phthalate (DBP) CAS: 84-74-2 MW: 278.34 g/mol Log Kow: 4.45		• Anti Foaming Agents • Cosmetics • Pesticides	• Teratogenic • Reproductive and developmental toxicity

Abbreviations: DEP, diethyl phthalate; DEHP, di(2-ethylhexyl) phthalate; DINP, diisononyl phthalate; BBP, benzyl butyl phthalate; DBP, di-n-butyl phthalate.



3.2. Physicochemical Properties Relevant to Migration

Longer alkyl chains mean lower water solubility and a stronger pull toward organic, fat-rich phases. This is not a subtle effect; oils, dairy products, and fatty meats take up phthalates with almost no resistance [3]. What actually lets a phthalate escape from plastic in the first place has more to do with what is missing chemically than what is present: there is no covalent bond holding it to the polymer. It just sits there, dissolved, held loosely by weak physical forces. Heat it, put it next to fatty food, let it sit on a shelf for months, and it starts to migrate out [3, 6]. A molecule abundant, weakly held, and fat-loving does not really need much of a push to end up in the food around it.

4. SOURCES AND MIGRATION PATHWAYS OF PHTHALATES IN FOODS

Three routes bring phthalates into food: migrate out of packaging, leaching from machinery handles food during manufacture, and entering the food chain through contaminated soil, water, and air. Which route dominates depends on the product, and each is examined below (Figure 1).

4.1. Food Packaging Materials

Packaging is usually the first suspect when phthalates turn up in food, and with good reason. These compounds are blended into PVC and related polymers as plasticizers, but they are never locked into the material; they sit between the polymer chains and escape whenever conditions allow. Fatty food, warm storage, and long shelf life all push in the same direction, moving molecules from the packaging into the very product it is meant to protect [10]. That is why edible oils, dairy products, and other lipid-rich foods generally carry higher phthalate loads than aqueous ones [11]. The type of contact material matters as well. Plastic wraps, multilayer films, caps and closures, gaskets, adhesives, and printing inks have all been identified as phthalate sources in food-contact applications, and recycled plastics introduce a further complication because their composition is difficult to control [6]. Regulators have struck several of the most problematic compounds from authorized lists, yet migration from packaging continues to be a measurable route of dietary exposure in practically every country where it has been studied [6, 10].

4.2. Food Processing Equipment

Packaging is not the only contact point. On the processing line, food runs through flexible tubing, crosses conveyor belts, passes seals and gaskets, and sits in storage tanks, many of which contain plasticized components. A single contact may last only minutes, but the number of contacts adds up, and several studies suggest that equipment can contribute as much phthalate to the finished product as its packaging does, occasionally more [11]. Industrial conditions favor release: elevated temperatures, long production runs, and repeated contact with the same plasticized surfaces all work in that direction. Dairy lines, beverage plants, and edible-oil refineries have been singled out as particularly susceptible [12].

Wear adds its own contribution. Aging gaskets, aggressive cleaning agents, and mechanical stress on plastic parts gradually free the additive from the polymer, which means a line that tested clean when installed may contaminate years later. Routine monitoring and timely replacement of worn components remain the simplest countermeasures available during manufacturing.

4.3. Environmental Contamination

The third route begins long before harvest or manufacture. Decades of intensive plastic use have left phthalates distributed through soil, water, air, and sediments across the globe [13]. Crops take them up from contaminated soil and irrigation water, sewage sludge spread on fields adds a further input, and atmospheric deposition completes the picture [13]. Residues in fruits, vegetables, and cereals are one result. Another is less direct: feed contaminated in this way carries the compounds into livestock, and measurable levels reappear in milk, eggs, and meat [12]. Plastic debris has made the situation worse. Fragmenting waste and microplastics release phthalates continuously as they weather, so environments that received plastics decades ago are still supplying fresh contamination [12, 13]. Unlike packaging or equipment, this route cannot be corrected by changing a material specification; it calls for broader environmental management, which is one reason the occurrence data presented in the next section vary so widely between regions.

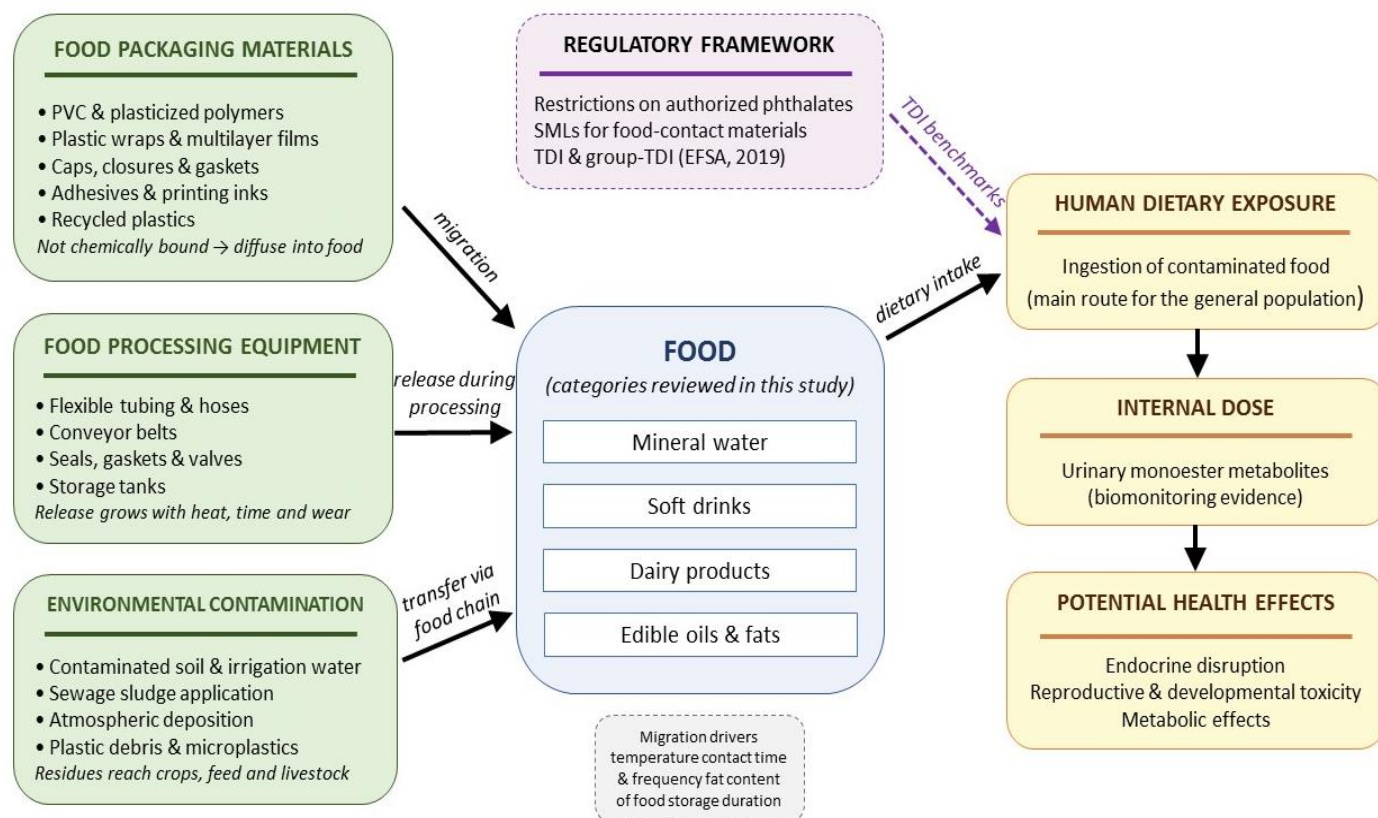


Figure 1. Main sources and migration pathways of phthalates into foods and the subsequent human dietary exposure. Solid arrows show the transfer of phthalates along the food chain; the dashed arrow indicates that estimated intake is benchmarked against tolerable daily intake (TDI) values established by regulatory frameworks, which also restrict the phthalates authorized for use in food-contact materials and set specific migration limits (SMLs) for them.

5. OCCURRENCE OF PHTHALATES IN MAJOR FOOD CATEGORIES

The pathways described above leave measurable residues. This section assembles what analytical surveys have found in the four food categories chosen for detailed treatment: mineral water, soft drinks, dairy products, and edible oils and fats. Because study quality in this field depends heavily on how contamination is kept out of the analytical procedure, a brief note on methods comes first. Table 2 at the end of the section summarizes representative studies, and Figure 2 compares typical concentration ranges between categories.

5.1. Analytical Methods and Quality Considerations

Nearly all occurrence data in this field rest on gas chromatography–mass spectrometry (GC-MS), usually after solvent extraction and a cleanup step, though liquid chromatography–tandem mass spectrometry is gaining ground. Headspace solid-phase microextraction coupled to GC-MS has been applied to milk to avoid solvent background altogether [14], and tandem MS in various configurations has served the analysis of olive oil [15]. Whatever the instrument, the analytical difficulty is the same: phthalates are everywhere in the laboratory. They leach from pipette tips, tubing, and solvent caps, so a careless blank can easily contain more analyte than the sample itself. Careful laboratories therefore work in glass rather than plastic, bake it before use, and run procedural blanks with every batch [11].



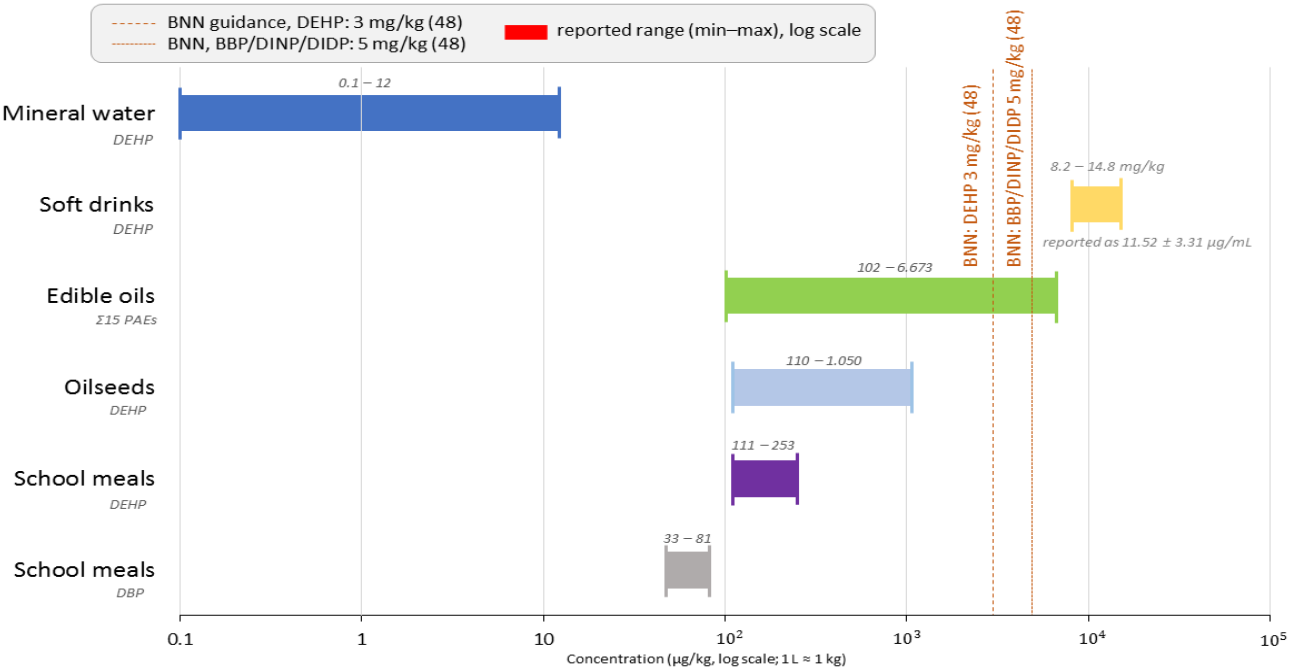


Figure 2. Reported concentration ranges of phthalates in the food categories reviewed (logarithmic scale; 1 L ≈ 1 kg). Each bar spans the minimum and maximum values reported in the study cited at the left, and the compound quantified is indicated for each row. Dashed lines show the guidance values of the German association for natural foods (BNN) for edible oils: 3 mg/kg for DEHP and 5 mg/kg for BBP, DINP, and DIDP.

Table 2. Representative studies on phthalate occurrence in foods and food-contact materials published between 2013 and 2026.

Year	Country/Region	Food Matrix	Main Phthalates	Analytical Method & Key Findings	Ref.
2013	Belgium	Milk chain (farm to retail)	8 PAEs (incl. DIBP, DBP, DEHP)	GC-MS; Milking tubing and packaging steps elevated residues; raw milk at farm carried minimal contamination	[40]
2015	Turkey	Six edible oil types	DEHP, DBP, others	GC-MS; 0.10–6.7 mg/L depending on lipid type; PET-bottled oils exhibited highest migration levels	[42]
2016	India	Bottled water, bovine milk	Multiple PAEs	GC-MS; Container leaching and environmental carryover; detectable in nearly all plastic-bottled retail milk	[31]
2016	China	Vegetable oils and oilseeds	DEHP, DBP	GC-MS; 0.02–1.05 mg/kg across 28 samples; residues derived from oil extraction, pressing equipment, and packaging	[43]
2018	Russia	Milk in polyethylene film	DMP, DBP, DEHP, BBP	GC-MS; Contact migration from PE pouches; detected in 70% of commercial pouch samples	[39]
2021	China	Bottled drinks	15 PAEs	LC-MS/MS; Beverage acidity accelerated leaching; soft drinks median Σ15 PAEs ≈ 5× higher than mineral water	[32]
2025	International (systematic review)	Packaging–food migration	Phthalates, bisphenols, PFAS	Systematic review; Polymer food-contact materials confirmed as predominant dietary migration vectors	[11]
2026	Spain/Italy	Fresh packaged fish	DEHP, DEHA	GC-MS/MS; Migration from packaging trays/films scaled with retail shelf-life, temperature, and fish lipid content	[53]

Abbreviations: PAEs, phthalate acid esters; DEHP, di(2-ethylhexyl) phthalate; DBP, di-n-butyl phthalate; DEHA, di(2-ethylhexyl) adipate.

Robust quality assurance further requires the mandatory incorporation of stable isotope-labeled internal standards (e.g., deuterated or ^{13}C -labeled analogues) spiked prior to extraction to correct for analyte recovery losses and matrix-induced ionization effects during MS detection. Furthermore, method validation protocols must explicitly report matrix-matched limits of detection (LOD) and quantification (LOQ), recovery rates (benchmarked to acceptable 70–120% thresholds), and procedural blank subtractions using pre-tested phthalate-free solvents and reagents [6, 12]. A methodology for distinguishing phthalates that migrate from packaging from those picked up elsewhere has been developed for exactly this purpose [16]. Comparisons between studies should be read with these caveats in mind, since detection limits and cleanup procedures differ from one laboratory to the next.

5.2. Mineral Water

Bottled water has grown into a huge market over the past three decades, first in North America and Europe and then across the developing world [17]. Most of it now travels in polyethylene terephthalate (PET) bottles, which are cheaper to produce than glass, although PVC was common earlier [18, 19]. PET is made from ethylene glycol and terephthalic acid under antimony catalysis, and residues of monomers, cyclic oligomers, and catalyst-derived antimony can transfer into bottled water [20–22]. Furthermore, phthalate plasticizers from printing inks and surrounding materials can migrate into packaged products [23], and under extreme storage or use conditions these compounds appear in the water as well [24]. Acetaldehyde formation remains a separate, better-known defect of PET, since it alters taste without posing the same toxicological questions [18]. Importantly, ortho-phthalates are not intentional constituents or functional additives in virgin PET resin, which attains requisite mechanical properties without plasticization; rather, trace phthalates detected in bottled beverages originate from auxiliary packaging components (plasticized cap liners, closure gaskets, printing inks, and labeling adhesives), post-consumer recycled PET (rPET) feedstock, or pre-bottling process contamination, rather than leaching from the PET polymer matrix itself [23, 41].

How much phthalate ends up in the bottle depends on circumstances. Temperature is the clearest driver: elevated storage temperatures (22 to 60 °C) and prolonged contact time have been linked to variable leaching patterns without a uniform linear trend across all phthalates [25, 26]. Sunlight acts similarly. In solar water disinfection (SODIS) experiments with used PET bottles exposed for 17 hours to full sunlight, Schmid and colleagues detected DEHP at a maximum concentration of 0.71 µg/L (DEHA 0.046 µg/L), in the same range as commercial bottled water, concluding that bottle origin and reuse history were more decisive than sunlight exposure or temperature [18]. Storage duration, pH, and bottle size matter too; the small 0.5 L containers showed the highest concentrations in one survey, presumably because their surface-to-volume ratio is larger [25]. Casajuana and Lacorte followed PET and polyethylene bottles over ten weeks at 30 °C and watched DEP, DBP, and DEHP climb from barely detectable starting levels [19]. Across surveys, DEHP has generally been the dominant compound, followed by DBP and diisobutyl phthalate (DIBP) [25], and tap water offers no escape: Montuori and colleagues found comparable levels across tap and bottled water samples alike [17].

There is a toxicological dimension as well. Water held in PET showed mutagenic potential in short-term assays, first reported by De Fusco and colleagues and later confirmed with the *Tradescantia* micronucleus test and Comet assay on leukocytes [20, 21]. More recent work shows the problem has not gone away. Phthalates were detected in bottled water from Tamil Nadu in India, alongside milk from the same region [22], and in the large Chinese market survey, mineral water carried PAEs at roughly five times lower levels than soft drinks, but far from zero [23]. Recent reviews of packaging migration reach the same conclusion for water as for other foods: the compounds keep arriving, mainly through the packaging itself [10].

5.3. Soft Drinks

Soft drinks sit in exactly the same containers as mineral water, yet they pick up more phthalates from them. Bošnjir and colleagues measured transfer 5 to 40 times higher into soft drinks than into mineral



water kept in identical plastic containers, and traced the difference to acidity: every soft drink they tested had a pH below 3, while the mineral waters stayed above 5 [24]. Acid clearly promotes migration.

The pattern repeats at market scale. In the Chinese survey mentioned above, the median total content of the fifteen PAEs measured in soft drinks exceeded that in bottled mineral water about fivefold, and the type of drink, not the bottle, was the main determinant [23]. A small HPLC study of plastic Coca-Cola bottles quantified five phthalates (DEP, DMP, DBP, DEHP, and DPhP) and reported DEHP at 11.52 ± 3.31 $\mu\text{g/mL}$, the highest of the set, with leaching increasing under heat and prolonged storage [25]. Whatever the exact figures, the direction is consistent: sugary, acidic beverages in PET are a more favorable medium for migration than water, worth remembering given how much of them children drink [12].

5.4. Dairy Products

Dairy is the category where processing equipment matters most. Milking machines, bulk tanks, and transfer lines all rely on PVC tubing, and DEHP can make up as much as 40% of that tubing by weight [26]. Milk passes through it warm, and because the plasticizer is not bonded to the polymer, some of it leaves with the product; several studies have documented this transfer from machine-milking equipment on dairy farms [14, 27].

Fat does the rest. Phthalates concentrate in the lipid phase of food, so cream, butter, and cheese accumulate more of them than low-fat dairy does [28]. The compounds also arrive through general contact with plasticized materials: PAEs have been reported in beverages, milk and milk products, meat and meat products, cooking oils, cereals, vegetables, and fruit alike [29].

Survey data back this up. Nearly all milk samples bought in plastic bottles contained PAEs in one study [22], and Zelenkin and colleagues found dimethyl phthalate (DMP), DBP, DEHP, and benzyl butyl phthalate (BBP) in 70% of milk samples packed in polyethylene film [39]. Perhaps the most instructive case is the milk-chain study of Fierens and colleagues, who followed eight phthalates from the farm to the

retail shelf [30]. Transport from farm to dairy plant added little; contamination rose where the milk met packaging, as the elevated DIBP, DBP, BBP, and DEHP levels in milk powder showed, and retail milk and cheese carried DIBP and DBP that the raw milk had not [40]. Children consume dairy in quantity, which makes these figures more consequential than their concentrations alone suggest [31].

5.5. Edible Oils and Fats

Oils combine every unfavorable property at once: they are fatty, they pass through plasticized equipment, and they are usually sold in plastic containers. Phthalates reach them from the raw material, from the production process, and from the packaging, by direct or indirect contact [32]. Their solubility in fat makes the outcome predictable, which is why fatty foods and olive oils have been analyzed so often [15]. The Turkish market survey of Sungur and colleagues shows the spread. Average concentrations ranged from 0.102 to 3.863 mg/L in virgin olive oil, 0.172 to 6.486 mg/L in olive oil, 0.501 to 3.651 mg/L in hazelnut oil, 0.457 to 3.415 mg/L in canola oil, 2.227 to 6.673 mg/L in sunflower oil, and 1.585 to 6.248 mg/L in maize oil; sunflower oil came out highest, virgin olive oil and hazelnut oil lowest, and oil sold in PET bottles carried more than oil in other containers [32]. Chinese oilseeds tell a similar story: in 28 samples, DEHP ranged from 0.11 to 1.05 mg/kg and DBP from 0.09 to 0.97 mg/kg, with the remaining PAEs between 0.02 and 0.39 mg/kg [33].

Against these figures stand the guidance values of the German association BNN, set in 2006 for the organic sector: 3 mg/kg for DEHP and 5 mg/kg for BBP, DINP, diisodecyl phthalate (DIDP), and related adipates [44, 45]. Parts of the ranges reported above sit close to, and in some cases above, those limits. Packaging takes a large share of the blame. Fankhauser-Noti and Grob quantified how much plasticizer migrates from the PVC gaskets of jar lids directly into oily foods [34], and migration from plastic containers in general has been confirmed for phthalates alongside bisphenol A and alkylphenols [35].

6. HUMAN EXPOSURE TO PHTHALATES

The food occurrence data reviewed above make one thing clear: complete avoidance of phthalate exposure is practically impossible. Because these compounds are not chemically bound to the products that contain them, they slowly escape into food, air, dust, and water. Biomonitoring studies confirm that this steady background release leads to measurable internal doses in virtually everyone examined.

6.1. Biomonitoring Evidence

Phthalates are absorbed, metabolized, and excreted within hours, which makes urinary mono-ester metabolites the standard markers of recent exposure. Measurements in large population samples show that exposure is nearly universal. In the USA, metabolites of eight major phthalates were found in 89 to 98% of the individuals tested [7]. Detection is not limited to one or two compounds either. Mono-(3-carboxypropyl) phthalate (MCPP), a metabolite shared by several high-molecular-weight parent compounds, appeared in almost every urine sample analyzed from pregnant women in NHANES cohorts [48], and elevated urinary phthalates have also been linked to metabolic alterations including diabetes among adult women in the same survey [47]. Children show similar detection rates, so exposure evidently starts early in life and continues at every age [7].

6.2. Routes of Exposure

Humans take up phthalates by several routes. The most obvious one is ingestion of contaminated food and water, but inhalation of indoor air and dust, skin contact with personal-care products, and medical exposures matter as well. Hospitalized patients can receive considerable doses intravenously, since phthalates leach from blood-storage bags, tubing, and enteral nutrition formulas. For the general population, however, diet remains the main source [3]. The relative weight of each route shifts with lifestyle and with the compound in question, because LMW and HMW phthalates occur in different product groups. One further difficulty deserves mention. Phthalate concentrations reported for the same food often differ widely between studies, and much of the older data no longer describes current exposure conditions [3].

6.3. Estimated Dietary Intake and Comparison with TDI

The central question for food safety is how much phthalate consumers actually ingest and how this amount compares with toxicological reference values. Methodologically, analytical occurrence (contaminant concentration in food) translates into dietary exposure only when integrated with consumption rates and body weight, whereas health risk is determined by benchmarking this exposure against toxicological hazard thresholds. The most recent comprehensive answer comes from the 2019 re-evaluation by the European Food Safety Authority (EFSA). The Panel confirmed the tolerable daily intakes (TDIs) set in 2005, namely 0.01 mg/kg bw per day for DBP, 0.5 for BBP, 0.05 for DEHP, and 0.15 mg/kg bw per day for DINP and DIDP, and added a group-TDI of 50 µg/kg bw per day, expressed as DEHP equivalents, to cover the joint action of DBP, BBP, DEHP, and DINP [36]. Aggregate dietary exposure to these four phthalates was estimated at 0.9 to 7.2 µg/kg bw per day for average consumers and 1.6 to 11.7 µg/kg bw per day for high consumers, which corresponds to at most 23% of the group-TDI. DIDP was not included in the group-TDI; its dietary exposure stays below 0.1 µg/kg bw per day, well under its own TDI of 150 µg/kg bw per day [36].

National assessments paint a comparable picture, although intake clearly depends on dietary habits. Among Chinese adults, ESTIMATED dietary intakes ranged from 0.02 µg/kg bw per day for benzyl butyl phthalate to 1.6 µg/kg bw per day for DEHP. Food, however, contributed less than 10% of total exposure to several of the low-molecular-weight compounds, which means non-dietary sources carry a large share of the overall burden in that population [29]. Children are a separate concern. In an assessment of school meals, DEHP and DBP intakes from cooked and packed lunches alone amounted to 12 to 18% of the EFSA TDI for DEHP and 30 to 50% of that for DBP in nursery and primary school children [37].

Overall, while current dietary intakes remain within tolerable limits (accounting for up to 23% of the group-TDI in high consumers, indicating a margin of safety for food intake alone), true risk characterization entails notable uncertainties; these



include unquantified non-dietary aggregate exposures, vulnerable developmental exposure windows, and real-world co-exposure to non-phthalate anti-androgens. Each of these calculations also treats one compound at a time, while real exposure involves mixtures whose combined effects are still poorly understood. The health consequences of this continuous, low-level exposure are examined in the next section.

7. HEALTH EFFECTS OF PHTHALATE EXPOSURE

The occurrence and exposure data assembled in the previous sections acquire their full weight only when set against what these compounds do inside the body. Phthalates belong to the endocrine-disrupting chemicals, and much of the toxicological work of the past two decades has tried to establish at which doses, in which tissues, and through which mechanisms their hormonal activity becomes harmful. This section reviews that evidence, with an eye on the dietary doses described in Section 6.

7.1. Mechanisms of Endocrine Disruption

Phthalates do not act on one receptor or one pathway; they interfere with the endocrine system at several points at once, and their metabolites matter more than the parent compounds. MEHP, the main metabolite of DEHP, binds the androgen receptor with an affinity approaching that of testosterone and competes with the natural hormone for the same binding site [38]. A second line of activity runs through the peroxisome proliferator-activated receptors, PPAR α and PPAR γ , which govern lipid metabolism, cell differentiation, and steroidogenesis in reproductive tissue. Experimental work with DEHP has connected activation of these receptors to mitochondrial dysfunction and apoptotic cell death in gonadal tissue [12]. What makes this receptor-level activity troubling is the dose. Effects appear at concentrations well below those that damage organs outright, and that is what separates an endocrine disruptor from a conventional toxicant.

7.2. Reproductive and Developmental Toxicity

The male reproductive tract takes the heaviest burden, and the developing organism more than the adult. In rodents given DEHP and structurally similar

phthalates during pregnancy, male offspring show a set of changes that has come to be called testicular dysgenesis syndrome: shorter anogenital distance, undescended testes, hypospadias, and poorly functioning Leydig cells [12, 38]. Adult animals and adult men respond differently. Chronic exposure has been linked to lower sperm concentration and motility, more sperm DNA damage, and falling circulating testosterone [4]. While rodent bioassays demonstrate biological plausibility under controlled experimental dosing, human epidemiological cohort studies primarily reveal statistical associations rather than proven direct causation, as residual confounding and mixed environmental co-exposures complicate definitive risk attribution [4]. Two groups deserve particular protection. The fetus and the newborn pass through developmental windows in which small hormonal perturbations can leave lasting marks, which is why pregnant women and young children are treated as the priority populations in dietary risk assessment [7].

7.3. Metabolic and Obesogenic Effects

A separate body of work asks whether phthalates influence body weight and metabolism. These compounds have been labeled obesogens, meaning chemicals that promote fat-cell formation and disturb energy balance. In children, dietary exposure to phthalates and bisphenol A has been associated with early-onset obesity and with precocious puberty, two conditions that often appear together and plausibly share endocrine mechanisms [5]. Candidate pathways include interference with thyroid hormone signaling, PPAR γ -driven adipogenesis in preadipocytes, and impaired insulin sensitivity. The same review is careful to note that results differ between studies, probably because exposure timing, dose, and population characteristics vary, so the obesogen hypothesis remains plausible rather than settled [5].

7.4. Hepatotoxicity and Other Systemic Effects

Phthalate metabolites also reach tissues outside the reproductive axis. They accumulate in erythrocytes, where they generate oxidative stress and can trigger eryptosis, a premature form of red-cell death [4]. The liver responds as well. Altered liver enzyme profiles have been reported in exposed pop-

ulations, and high-dose animal experiments produce hepatocellular changes typical of peroxisome proliferation. Whether the animal liver findings say anything about people eating contaminated food is contested, since the effective doses exceed realistic dietary intakes by a wide margin [12].

7.5. Relevance to Food Safety

For food safety, the mechanism matters less than the arithmetic. The exposure estimates of Section 6 show high consumers approaching a quarter of the group-TDI and school meals consuming a sizable fraction of a child's daily allowance, all from a source that individuals cannot see, taste, or choose to avoid. The toxic effects summarized in Table 1 attach to compounds that reach food through packaging, equipment, and the environment, the same routes traced in Section 4. Exposure of this kind is continuous, low-dose, and involuntary. That combination is what turns migration data into a public health question, and it explains why even modest reductions in migration, particularly for pregnant women and children, are worth pursuing at the regulatory level examined in the next section.

8. REGULATORY FRAMEWORKS FOR PHTHALATES IN FOOD-CONTACT MATERIALS

Regulators approach phthalates from two directions at once. The toxicological side asks how much a person may ingest without harm and answers with a tolerable daily intake. The food-contact side converts that allowance into specific migration limits (SMLs), which cap the concentration a material may transfer into food or a food simulant. Both instruments exist in the major food-producing regions, though not always with the same scope or the same numbers, and the gaps between them are where uncertainty concentrates.

8.1. European Union

The EU moved furthest, and its system is anchored in the EFSA assessment discussed in Section 6. The 2019 update confirmed the TDIs of 0.01 mg/kg bw per day for DBP, 0.5 for BBP, 0.05 for DEHP, and 0.15 for DINP and DIDP, and added a group-TDI of 50 µg/kg bw per day, expressed as DEHP equivalents, to account for the joint action of four of these com-

pounds [36]. Binding limits follow from this evaluation. Commission Regulation (EU) No 10/2011 on plastic food-contact materials sets SMLs of 1.5 mg/kg for DEHP, 0.3 for DBP, 30 for BBP, and 9 mg/kg for DINP and DIDP [54]. A subsequent amendment in 2020 (Commission Regulation (EU) 2020/1245) narrowed the field further: DEHP, DBP, BBP, and DIBP are no longer permitted in general-purpose plasticized packaging for infant and young-child foods, and several other applications were withdrawn [54].

8.2. United States

The American picture is messier. Phthalates enter the FDA's food-additive framework (21 CFR) as plasticizers in defined food-contact uses, and the agency has been pruning that list rather than building a comprehensive scheme. In 2022 it revoked the authorizations of more than two dozen phthalates on the grounds that manufacturers had abandoned their use, leaving a smaller set of uses in place pending further review [55]. Outside food, the Consumer Product Safety Improvement Act has banned six phthalates above 0.1% in toys and childcare articles since 2008, which shows the appetite for restriction exists in other product categories even when food-contact rules lag [6].

8.3. Other Jurisdictions

Canada restricts the principal phthalates in children's products and assesses food-contact submissions through its own risk-review process, with limits broadly comparable to the EU list. China's national standard GB 9685-2016 permits a defined set of phthalates in food-contact materials and attaches migration limits to them, values that largely track the European numbers [39]. Japan reorganized its system in 2020, when the amended Food Sanitation Act replaced the older voluntary association standards with a positive list for food-contact plastics; phthalates appear on the list with conditions rather than being banned outright [39]. The differences sound technical, but they matter in trade: a formulation compliant in one market can breach the rules of another.

8.4. Alternative Plasticizers and Remaining Gaps

Restrictions created their own market. DINCH and DEHT, the two plasticizers most often



substituted for restricted phthalates, have grown rapidly in food-contact use, and toxicological information on them is still accumulating; their safety profiles rest on shorter exposure histories than those of the compounds they replaced [6]. DCHP and several other phthalates remain under evaluation without assigned limits (Table 3). Two further weaknesses deserve mention. Recycled plastics,

whose composition is inherently variable, are only partly covered by current authorization schemes, and import enforcement is thin, since compliance checks depend on national sampling budgets. A rule that exists on paper but escapes routine monitoring reduces migration only where inspectors look.

Table 3. Regulatory status of the major phthalates in food-contact materials across the European Union, the United States, Canada, China, and Japan.

Phthalate	European Union (Reg. EU 10/2011; EFSA 2019)	United States (FDA 21 CFR; CPSIA)	Canada (Food & Drugs Act; CCPSA)	China (GB 9685- 2016)	Japan (Food Sanitation Act, 2020)
DEHP	Group-TDI 50 µg/kg bw/day (sum); SML 1.5 mg/kg; Prohibited in infant food packaging	21 CFR §175–178 authorizations revoked (2022); CPSIA limit <0.1% in child products	Restricted in FCMs; CCPSA limit <1,000 mg/kg (0.1%) in toys/childcare	SML 1.5 mg/kg per GB 9685; Prohibited for fatty foods and infant contact	Positive list system (2020); Banned in PVC toys and fatty food-contact plastics
DBP	Allocated to Group-TDI (50 µg/kg bw); SML 0.3 mg/kg; Prohibited in infant packaging	Food-contact authorizations revoked (2022); CPSIA banned >0.1%	Restricted in food packaging; CCPSA limit <0.1%	SML 0.3 mg/kg; Prohibited for infant food contact materials	Positive list conditional use; Banned in flexible materials contacting oily foods
BBP	Allocated to Group-TDI (50 µg/kg bw); SML 30 mg/kg; Prohibited in infant packaging	Food-contact authorizations revoked (2022); CPSIA banned >0.1%	Restricted in food packaging; CCPSA limit <0.1%	SML 30 mg/kg per GB 9685 positive list	Conditional authorized use under Positive List System
DINP	Allocated to Group-TDI (50 µg/kg bw); SML 9 mg/kg (sum with DIDP)	Permanent CPSIA ban (>0.1% in child items); Limited indirect additive uses	Interim restriction in mouthed child items; Case-by- case review in FCM	SML 9 mg/kg; Authorized only for non-fat food- contact plastics	Restricted under Positive List; Prohibited for infant mouth contact
DIDP	Individual TDI 0.15 mg/kg bw/day; SML 9 mg/kg (sum with DINP)	Risk assessment by CPSC/FDA; Authorized in select repeat-use rubber/polymers	Subject to screening assessments; Regulated on risk basis	SML 9 mg/kg; Specific application restrictions apply	Positive list conditional authorized use
DCHP	Identified as SVHC; Evaluated under REACH Annex XIV; SML restricted	Authorizations revoked (2022); Included in cumulative priority assessments	Evaluated under Chemicals Management Plan (CMP)	Not listed on GB 9685-2016 positive list (unauthorized)	Restricted; Subject to positive list evaluation
Alternatives (DINCH, DEHT)	DINCH: SML 60 mg/kg; DEHT: SML 60 mg/kg; Under ongoing safety monitoring	Authorized for food- contact polymers via Food Contact Notifications (FCN)	Evaluated case- by-case via Food Directorate pre- market submissions	Listed in GB 9685- 2016 positive list with specified SMLs	Approved on Positive List for general plastic formulations

Abbreviations: SML, specific migration limit; TDI, tolerable daily intake; bw, body weight; DINCH, di(isononyl) cyclohexane-1,2-dicarboxylate; DEHT, di(2-ethylhexyl) terephthalate; DCHP, dicyclohexyl phthalate.

9. REMOVAL AND BIODETOXIFICATION OF PHTHALATES BY LACTIC ACID BACTERIA

Because regulatory limits and packaging changes cannot eliminate phthalates already circulating through food processing chains, several research groups have explored biological approaches to reduce contamination before food is consumed. Lactic acid bacteria (LAB) and common probiotic strains are logical candidates for this role. They carry Generally Recognized as Safe (GRAS) status, are already widely used in fermented milks, yogurts, and sourdoughs, and introduce no synthetic chemicals to the product. Rather than degrading the plasticizers through complex metabolic pathways, these bacteria act chiefly by physical biosorption: phthalate molecules bind directly to the bacterial cell envelope, lowering the concentration of free, bioaccessible toxicant available for systemic absorption during digestion [56, 57]. Here, a clear mechanistic distinction must be drawn between active enzymatic biodegradation (metabolic catabolism into mineralized end-products) and passive surface biosorption (physico-chemical entrapment by cell envelope components). Food-grade lactic acid bacteria utilized as fermentative starter cultures primarily execute the latter, whereas specific characterized probiotic strains additionally confer *in vivo* protection by maintaining mucosal barrier integrity, thereby reducing systemic absorption without requiring enzymatic cleavage of the diester [56, 57].

9.1. Cell-Wall Binding and the Role of Peptidoglycan

Most of the removal occurs directly at the bacterial cell surface. Zhao and colleagues investigated how *Lactobacillus (L.) acidophilus* takes up di-n-butyl phthalate (DBP) from aqueous solutions and found that the cell wall accounts for the bulk of the retention, with the peptidoglycan network providing roughly 81% of the total binding capacity [58]. The sequestration does not depend on active bacterial metabolism; heat-killed cells take up nearly as much phthalate as living cultures, confirming that the mechanism is physical adsorption rather than an enzymatic process [56, 58]. Molecular dynamics

simulations showed that hydrophobic interactions drive the binding, supported by van der Waals forces and hydrogen bonds between the ester side chains of the phthalate and exposed functional groups (O–H, N–H, C–N, and C–O) on the peptidoglycan matrix [58]. Diethyl phthalate and dioctyl phthalate follow similar binding patterns, with longer, more hydrophobic alkyl chains generally promoting stronger attachment. The strains, experimental matrices, binding efficiencies, and operational conditions investigated across these studies are systematically summarized in Table 4.

9.2. Contribution of Surface Exopolysaccharides

Peptidoglycan is not the only bacterial component involved. Many lactic acid bacteria secrete an outer layer of exopolysaccharides (EPS) that alters how the cell interacts with lipophilic compounds. Fan and colleagues evaluated DBP removal by *Lactiplantibacillus plantarum* CGMCC18980, a strain capable of adsorbing 58.6% of ambient DBP [59]. When the EPS layer was stripped enzymatically, the adsorption percentage dropped by almost half, whereas removing outer membrane proteins left binding largely unaffected [59]. Electron micrographs revealed a distinct surface coating forming on the bacterial exterior following exposure, and infrared spectroscopy showed noticeable vibrational shifts in C=O and C–O bands of the carbohydrate fraction [59]. Surface charge measurements showed no significant difference before and after binding, indicating that electrostatic attraction plays a minor role compared to hydrophobic entrapment [59]. Strains tested with di(2-ethylhexyl) phthalate (DEHP) exhibit comparable behavior, where adsorption follows Langmuir-type isotherms with defined, saturable surface binding sites [60].

9.3. Behavior in Food Matrices and In Vivo Effects

In real foods and the digestive tract, microorganisms encounter both intact parent diesters and the monoester metabolites produced when phthalates hydrolyze. Mono-(2-ethylhexyl) phthalate (MEHP) and monobutyl phthalate (MBP) are more water-soluble than parent DEHP or DBP, but they are also more toxic. Zhu and colleagues



screened dairy *Lactobacillus* strains against these monoesters and found that binding was strongest under acidic conditions (pH 3 to 5), the range typical of fermented milks and gastric juice [61]. The resulting complexes remained stable under simulated stomach and intestinal conditions, indicating that bacteria can hold the trapped molecules through the gut rather than releasing them as pH shifts [56, 61].

Animal studies test whether this retention translates into lower tissue burdens. Xiao and colleagues administered DEHP, DBP, and DINP to rats alongside a multi-strain probiotic formulation (*Lactiplantibacillus plantarum*, *Limosilactobacillus reuteri*, and *Bifidobacterium bifidum*) and tracked toxicant excretion [62]. Rats receiving the probiotic excreted substantially more parent phthalates and monoesters in their feces, accumulated less plasticizer in the liver, kidney, and testes, and showed

lower serum markers of oxidative stress and tissue injury [62]. Probiotics also appear to help by maintaining the gut barrier itself; by supporting epithelial tight junctions and suppressing local inflammation, they reduce the ease with which unbound phthalate crosses the intestinal wall into circulation [57]. Nonetheless, while in vitro simulations and rodent bioassays provide compelling proof-of-concept for biosorptive sequestration, validated commercial applications in food processing lines and human clinical trials are currently lacking; consequently, probiotic-mediated mitigation must be framed as a promising biological possibility rather than an established clinical or industrial reality [56, 57, 62].

Table 4. Summary of food-grade lactic acid bacteria (LAB) and characterized probiotic strains investigated for phthalate biosorption and in vivo mitigation.

Microbial Strain	Target Phthalate	Tested Matrix/System	Removal Efficiency/Outcome	Operational Conditions	Proposed Mechanism	Evidence Level & Ref.
<i>L. acidophilus</i>	DBP	Aqueous solution	~81% cell-wall retention	37 °C, pH 6.0; viable and heat-killed cells	Hydrophobic entrapment within peptidoglycan network	In vitro model [58]
<i>Lactiplantibacillus plantarum</i> CGMCC18980	DBP	Model broth	58.6% removal efficiency	37 °C; Langmuir-type adsorption isotherm	Surface exopolysaccharide (EPS) layer binding	In vitro model [59]
<i>Lactobacillus</i> spp.	DEHP	Aqueous medium	Up to 65% sorption	pH 4.0–7.0; concentration-dependent saturation	Physicochemical cell envelope biosorption	In vitro model [56]
Dairy <i>Lactobacillus</i> strains	MEHP, MBP	Fermented milk / simulated GI	Up to 70% monoester complexation	Acidic pH (3.0–5.0); stable through simulated digestion	Cell-wall functional group complexation	In vitro digestion [61]
Probiotic formula (<i>L. plantarum</i> , <i>L. reuteri</i> , <i>B. bifidum</i>)	DEHP, DBP, DINP	Rat in vivo model (oral gavage)	Significantly accelerated fecal clearance; reduced organ bioaccumulation	Daily oral co-administration over test period	Intestinal barrier preservation & luminal sequestration	In vivo rodent model [62]

Abbreviations: LAB, lactic acid bacteria; DBP, di-n-butyl phthalate; DEHP, di(2-ethylhexyl) phthalate; DINP, diisononyl phthalate; MEHP, mono(2-ethylhexyl) phthalate; MBP, monobutyl phthalate; EPS, exopolysaccharides; GI, gastrointestinal.

10. RESEARCH GAPS AND FUTURE PERSPECTIVES

Writing this review made the holes in the literature as visible as its contents. Several of them deserve to be named, because each one limits what food safety authorities can currently say with confidence. The first concerns the age and patchiness of the occurrence data. For mineral water, the bulk of the published measurements predates 2015, even though formulations, storage practices, and consumption patterns have all changed since [17, 40]. Surveys from low- and middle-income countries are scarce, so the global picture rests disproportionately on a handful of European, North American, and Chinese studies [22, 23]. Where data are old or geographically thin, exposure assessments inherit both weaknesses.

A second gap is analytical. As Section 5.1 showed, phthalates contaminate the laboratory as readily as the food, and procedural blanks, detection limits, and cleanup procedures still differ enough between laboratories to make quantitative comparisons across studies unreliable [11]. Standardized, contamination-controlled protocols, ideally with certified reference materials, would do more to improve this field than any single new survey.

Third, exposure assessment remains largely single-compound. The group-TDI introduced by EFSA in 2019 was a first step toward cumulative risk assessment, yet most published intake estimates still treat each phthalate in isolation. Real diets deliver mixtures of phthalates alongside bisphenols and other plastic-derived chemicals, and the Chinese data discussed in Section 6 warn that food may not even be the dominant source for some compounds [29]. Cumulative, multi-source assessment is methodologically demanding, but without it the tolerable intakes now in force may not describe the risk consumers actually carry.

Fourth, migration has mostly been measured under standardized test conditions rather than real ones. Household storage involves fluctuating temperatures, months of shelf time, and repeated opening and closing, and the few studies that approximated these conditions found migration continuing to rise with storage time [41]. Systematic

migration data under realistic use scenarios are needed before packaging choices can be optimized honestly.

The fifth gap follows from the fourth. Restrictions on the older phthalates pushed manufacturers toward substitutes such as DINCH and DEHT, whose toxicological records are short and whose metabolites are only beginning to appear in biomonitoring programs [6]. History argues for caution here: substitutes for other industrial chemicals have, more than once, carried problems of their own. Independent long-term studies of the replacements should precede, not follow, their market saturation. Finally, regulation itself remains fragmented. As Table 3 illustrates, a plasticizer withdrawn in one jurisdiction may still be authorized in another, and enforcement depends on national monitoring budgets [42, 43]. Harmonized limits and shared monitoring data would close part of this gap, and they would also give researchers what the field most conspicuously lacks: comparable, current, and openly reported occurrence data against which any progress can be measured.

11. CONCLUSION

Phthalates enter the food chain through multiple contact points: polymer packaging on the shelf, tubing and gaskets on the processing line, and environmental residues that contaminate agricultural commodities long before harvest. Occurrence surveys across major food categories reflect the lipophilic chemistry of these compounds. Lipid-rich items such as edible oils and dairy products accumulate the heaviest loads, while aqueous matrices like bottled water and soft drinks demonstrate that acidity, contact surface area, and prolonged storage can also drive measurable migration.

Although individual residues in most surveys fall below existing specific migration limits, the overall exposure picture warrants concern. High consumers and young children eating packaged diets absorb a meaningful fraction of established tolerable daily intakes, particularly when evaluated against cumulative group-TDI standards. Regulatory restrictions have curtailed several high-production phthalates, pushing industry toward substitutes such



as DINCH and DEHT; however, these chemical replacements enter commerce ahead of thorough chronic toxicity assessments, and they do nothing to address contamination already present in food equipment and the wider environment.

In this context, biological mitigation by food-grade lactic acid bacteria and characterized probiotic strains represents a promising investigational intervention. Physical biosorption mediated by the peptidoglycan matrix and cell-surface exopolysaccharides enables strains such as *L. acidophilus* and *L. plantarum* to sequester parent diesters and reactive monoesters (e.g., MEHP) in model matrices. Furthermore, rodent bioassays indicate that this luminal entrapment can accelerate fecal excretion, preserve epithelial barrier integrity, and attenuate target-organ bioaccumulation. However, these benefits currently remain at the preclinical proof-of-concept stage. Translating laboratory biosorption into validated dietary protection will require demonstrating binding stability across real-world commercial processing conditions, establishing human clinical efficacy, and harmonizing micro-biological strategies with cumulative regulatory frameworks.

12. DECLARATIONS

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12.5. Ethical Considerations: All ethical principles were adhered to in conducting and writing this article.

12.6. Data availability: In accordance with the principles of transparency and open research, we declare that all data and materials used in this study are available upon request.

12.7. Using Artificial Intelligence chatbots:

No AI chatbot has been used in this study.

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